RISK FACTOR ANALYSIS AND ANGIOGRAPHIC PROFILE IN YOUNG MYOCARDIAL INFARCTION

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CERTIFICATE

This is to certify that the dissertation entitled **"RISK FACTOR ANALYSIS AND ANGIOGRAPHIC PROFILE IN YOUNG MYOCARDIAL INFARCTION"** is the bonafide original work of **DR. E. ARUNACHALAM** in partial fulfillment of the requirements for D.M. Branch-II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2010. The period of post-graduate study and training was from July 2007 to July 2010.

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DECLARATION

I , Dr. E. ARUNACHALAM, solemnly declare that this dissertation entitled, **"RISK FACTOR ANALYSIS AND ANGIOGRAPHIC PROFILE IN YOUNG MYOCARDIAL INFARCTION"** is a bonafide work done by me at the Department of Cardiology, Government Stanley Medical College and Hospital, Chennai during the period 2007 – 2010 under the guidance and supervision of the Professor and Head of the Department of Cardiology of Government Stanley Medical College and Hospital, **Professor G. KARTHIKEYAN**, **M.D., D.M.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-II) in CARDIOLOGY.**

Place: Chennai **Dr. E. ARUNACHALAM**

Date:

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CONTENTS

INTRODUCTION

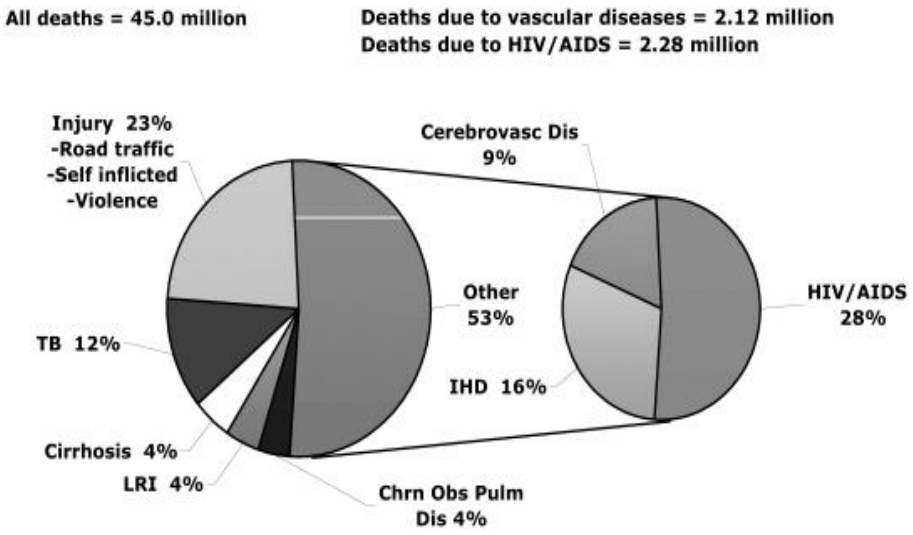
Cardiovascular disease (CVD) has become a major clinical and public health problem. South Asian countries, namely India, Pakistan, Sri Lanka, Bangladesh and Nepal, not only represent a quarter of the world's population but also contribute to the highest proportion of CVD burden when compared with any other regions globally. This population carries the increased risk even if they migrate to other countries and have increased mortality due to CVD at a younger age in comparison to the local population the risk remains high in Indians regardless of whether they have immigrated to the west or live in their native countries. They have the highest risk of developing CVD at a younger age in comparison to other ethnic groups of particular concern to India is not only the high burden of cardiovascular diseases (CVDs), but also the effects of these diseases on the productive workforce aged 35–65 years. Heart diseases are rising in Asian Indians 5–10 years earlier than in other populations around the world. The mean age for first presentation of acute myocardial infarction in Indians is 53 years. Coronary artery disease (CAD) that manifests at a younger age can have devastating consequences for an individual, the family, and society.

Projections show that CVD has reached epidemic proportions in many developing countries. In India, mortality attributable to CVD is expected to rise by 103% in men and by 90% in women from 1985 to 2015[3]. More importantly, the disease catches Indians young. The risks are explained on the basis of traditional and non-traditional risk factors, thereby suggesting that reducing these risk factors can ultimately lead to decreased burden of CVD in this population. Risk for developing CVD emerges at a relatively young age in the Indian population, and women have a risk similar to that of men. Women also have greater prevalence of hypertension and left ventricular hypertrophy (LVH). We discuss the conventional risk factors prevalent in young Indian CAD patients. We have restricted ourselves to well established CAD-associated risk factors, as these are the immediate targets for cardiac event risk reduction worldwide.

Rising prevalence of cardiovascular diseases globally:

CVDs are no longer confined by geographical area or by age, sex, or socioeconomic boundaries. Heart disease has already reached epidemic proportions in poorer countries. Of the 45.0 million adult deaths reported worldwide in 2002, three-quarters (32 million) were due to noncommunicable diseases. Except in Africa, noncommunicable diseases outnumbered communicable diseases in all WHO regions worldwide. In Southeast Asia alone, 7 423 000 deaths were due to

noncommunicable diseases as compared with 5 730 000 deaths related to communicable diseases in the year 2002. Globally, ischemic heart disease (IHD) was the leading killer in the age group ≥60 years, and, with 1 332 000 deaths in adults.



Leading causes of death worldwide in 2002 (age 15–59 years). **Abbreviations:** Cerebrovasc Dis, cerebrovascular disease; Chrn Obs Pulm Dis, chronic obstructive pulmonary disease; IHD, ischemic heart disease; LRI, lower respiratory infection.

With 6.8% and 5.0% of disability-adjusted life-years (DALYs) lost, CAD and stroke were globally the second and third largest causes of disease burden in men aged 15 years and older in 2002. Even in women, CAD and stroke were the third and fourth main causes of DALYs lost worldwide.

Trends of cardiovascular diseases in India:

It is well known that the demographic transition in Western countries was accompanied by a decrease in deaths due to infectious diseases and increased mortality due to noncommunicable diseases. India is in the midst of such demographic transition. The average life expectancy at birth in India is 63.7 years, being 63.1 for males and 64.4 for females[4] , compared with the national average of 41.2 years in 1951–1961 However, this demographic transition has also led to an increase in the number of older people (aged ≥60 years), from 19.61 million in 1950 to 75.93 million in 2000[5]. The increase in life expectancy has brought a large section of the population to an age where CVD starts manifesting itself.

In India, CAD rates have increased during the last 30 years, whereas declining trends have been noticed in developed Western countries [6]. Reports on CAD in Indians from different parts of the world have shown that Asian Indians are at 3–4 times higher risk of CAD than

white Americans,6 times higher than Chinese, and 20 times higher than Japanese. [7]

The exact prevalence of CAD in India is difficult to estimate owing to the lack of a large prospective study. Absence of a centralized death registry for CVDs and irregularities in completion of death certificates also hamper estimation of the actual burden of CVD. However, various independent epidemiological studies [8,9] conducted in North India suggest that the prevalence of CAD has increased from 1% in 1960 to 10.5% in 1998 in the urban population. A higher prevalence of CAD, ranging from 11.0% to 14.2%, has been reported from South India [2]. In rural India, a twofold increase has been reported in the northern states [10,11]. A higher prevalence of 7.4% was observed in some parts of rural South India as long ago as in 1993 [12]. Taking into account the size of the Indian population, these prevalence rates, translated into figures, indicate that a large number of deaths can be attributed to CAD.

Premature CAD in Indians:

Of particular concern to India is not only the high burden of CVDs, but also the effects of CVD on the productive workforce aged 35–65 years.

- India topped the world with 1 531 534 cardiovascular disease-related deaths in 2002
- Median age of first heart attack in Indians is 53 years

- Incidence of CAD in young Indians is about 12%–16%, which is higher than any other ethnic group

Age-standardized estimates for disability-adjusted life-years lost due to CAD per 1000 population in India are three times higher than in developed countries [13,14 ,15]

The incidence of CAD in the young has been reported to be 12%–16% in Indians [13, 14]. Half of the CVD-related deaths (ie, 52% of CVDs) in India occur below the age of 50 years, and about 25% of acute myocardial infarction (MI) in India occurs under the age of 40 years .[16]

Heart diseases are occurring in Indians 5 to 10 years earlier than in other populations around the world [17]. According to the INTERHEART study, the median age for first presentation of acute MI in the South Asian (Bangladesh, India, Nepal, Pakistan, Sri Lanka) population is 53 years, whereas that in Western Europe, China, and Hong Kong is 63 years, with more men than women affected. Data from the Singapore Myocardial Infarction Registry from 1988 to 1997 for acute MI cases aged between 20 and 64 years also showed that men were four times more prone to these events than women [18]. The median age for presentation of first MI was higher in Asian women than in Asian men (58 and 54 years, respectively), a finding similar to that of the INTERHEART study. Among the three Asian populations, Chinese,

Malay, and Indian, the highest age-standardized incidence rates in both sexes are in Indians. The first MI attack occurs in 4.4% of Asian women and 9.7% of men at age less than 40 years, which is 2- to 3.5-fold higher than in the West European population and is third highest of all the regions studied worldwide. These studies carried out in India and other places suggest that Asians in general and Indians in particular are at increased risk of MI at a younger age (<40 years), irrespective of whether they have migrated to other countries or are resident Asians.

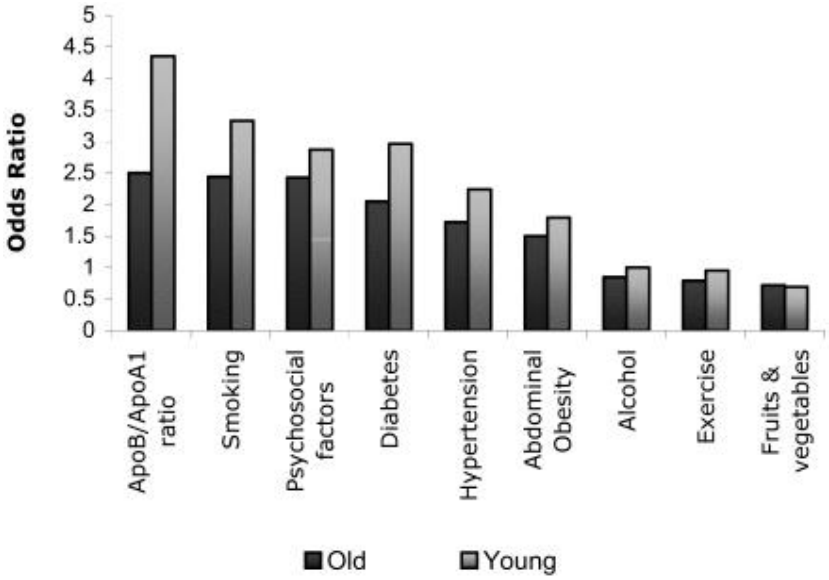
It must be emphasized that although the median age of presentation is higher in women, they are known worldwide to have poor prognosis compared with men [18,19,20]. Younger Asian women have worse survival at 28 days after acute MI [21]. The reasons for higher mortality in younger women are poorly understood and may be related to the presence of different risk factors in women, comorbidities, severity of infarction, and response to treatment. In view of the above discussion, it is imperative to ascertain the causes of the rising prevalence and emergence of CAD earlier in the life of Indians.

Risk factors for premature CAD in Indians:

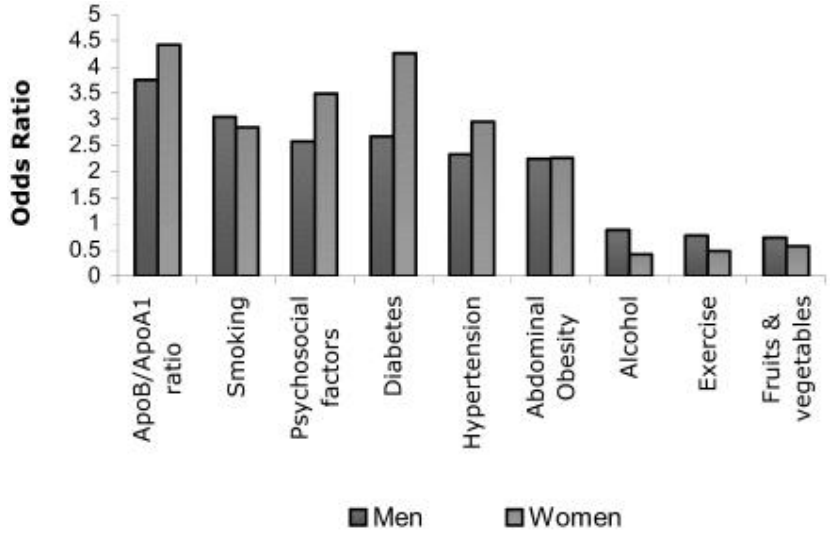
Most of the knowledge on CAD risk factors in different age groups is from studies carried out in the migrant Indian population [22,23] and these have their limitations.. For these reasons, it has been widely speculated that the observations in the migrant population may not hold

true for the Indian population. Therefore, we review here only those studies that were carried out in resident Indians, as this provides the true status of the prevalence of conventional CAD risk factors in this population.

Recently, the INTERHEART study established an association between conventional modifiable risk factors for MI in all regions of the world, including south Asia, and in both sexes and at all ages.



Odds ratio for myocardial infarction risk factors worldwide; old (>53 years) versus young (<53 years).



Odds ratio for myocardial infarction risk factors worldwide; men versus women.

In South Asians, apolipoprotein (Apo)B/ApoA1 (odds ratio [OR] 3.81) and smoking (OR 2.43) were the important risk factors, as in the rest of the world. However, hypertension (OR 2.89), abdominal obesity (OR 2.43), and diabetes (OR 2.48) had more severe effects in South Asia, whereas psychosocial factors had an OR of 2.15, compared with 2.67 worldwide. The INTERHEART study also showed that hypertension and diabetes were more important risk factors in younger Indian women than men. Earlier studies, mostly in Western populations, have also found an association of the above-mentioned risk factors with the development of CAD. For example, in the Prospective Cardiovascular Münster Heart Study (PROCAM), a large prospective study in men aged 35–65 years, eight variables that made an

independent contribution to risk of CAD were age, systolic blood pressure, LDL-C, HDL-C, triglycerides, diabetes mellitus, smoking, and family history of MI [1].

Fewer studies on epidemiological data from angiographically proven cases of premature CAD (≤40 years) in native Indians are available [24 ,25,26] Hyperlipidemia was found to be prevalent in young Indians with CAD in these studies. However, differences appear to exist between the lipid levels present in North and South Indian CAD patients and individuals without CAD. It appears that North Indians manifest the disease at lower levels of total cholesterol [27,28,29]. Also, a greater role can be attributed to total cholesterol and LDL-C in atherogenesis in the younger Indian population (≤40 years) with angiographically proven CAD. The lower HDL-C and higher triglyceride levels in both younger and older cases appear to be a hallmark of the Indian population . In the INTERHEART also, the highest population attributable risk (PAR) was abnormal lipids (ApoB/ApoA1 ratio) in both sexes. These studies indicate that abnormalities in lipid metabolism play an important role in development of CAD in young Indians. Also, compared with women, young Indian male patients have a slightly lower prevalence of hypertension and diabetes.

Smoking and low physical activity in Indians have been found to be prevalent in 20–39-year-old urban adults. The INTERHEART study

also observed that smoking was a greater risk factor in younger men than in women. The risk of CAD increased incrementally with smoking. The OR was 9.16 in individuals who smoked more than 40 cigarettes per day, compared with 1.38 in those smoking 1–5 cigarettes per day, indicating that there is no safe limit for smoking. Other epidemiological studies from India also suggest a greater association of smoking with CAD in younger individuals [30]. Furthermore, the prevalence of smoking in South Indian males (44.6%) and passive smoking in South Indian females (45.3%) has been reported to be significantly higher than in North Indians. Interestingly, smoking has not been found to be a significant risk factor in acute MI patients from rural parts of India. The patients from rural India, however, have elevated blood glucose and abnormal waist/hip ratio .

Another important independent risk factor for CAD in younger cases emerging out of Indian studies is family history of CAD . The INTERHEART study showed a PAR of 14.8% in younger versus 10.45% in older patients . Though addition of family history of CAD to other risk factors causes only a modest increase in PAR by 1% , it must be emphasized that modifiable physiological variables such as blood pressure, ApoB/ApoA1 ratio, serum cholesterol, and ab hemostatic factors, lipid metabolism, and other metabolic factors is warranted.

With rapid urbanization and industrialization, a nutritional transition is occurring in India. A continual increase in the prevalence of obesity is being seen. With the introduction of an era of refined foods, including refined flour (maida), sugar, and hydrogenated oils, the traditional high complex carbohydrate, high fiber, low-fat diet has been replaced by a diet rich in fats and readily absorbable simple sugars and low in minerals and dietary fiber, a scenario threatening to produce similar health hazards to those seen in well developed nations. It is, however, important to emphasize that this nutritional transition is not inevitable. Public health professionals need to develop strategies so that a healthier transition occurs.

Coronary Collateral Circulation:

Coronary collateral arteries serve as alternative conduits for blood flow in obstructive coronary heart disease (CHD).The assessment, pathogenesis, and therapeutic promotion of coronary collaterals have recently been described by Seiler.[1]

Collateral artery growth is mediated by arteriogenesis. Anatomically, collateral arteries may be either epicardial or intramyocardial and serve as contralateral or ipsilateral conduits .Myocardial blood flow is the product of epicardial coronary and collateral artery flow. Coronary occlusion during the acute stage of

myocardial infarction results from a complex interaction of factors including plaque fissure, platelet deposition, arterial spasm, and fibrin deposition. Spontaneous recanalization, which has also been documented, may result from resolution of spasm, breakdown of platelet deposits, and activation of the endogenous thrombolytic system. Collateral vessels were determined to be present if any segment of the infarct-related artery filled in any other than a continuous antegrade manner.

Attribute	Definition
Angiogenesis	Formation of new capillaries by sprouting from post-capillary venules
Arteriogenesis	The transformation of pre-existing arterioles into functional (muscular) collateral arteries with vasomotor properties
Collateral blood vessel	Collateral blood vessels are vascular connections linking parallel arteries without an intervening capillary bed

In recent years, coronary collateral circulation in the setting of ischemic heart disease and particularly in myocardial infarction has been subjected to special interest. Some degree of collateral circulation is

present at the onset of acute myocardial infarction (AMI) in nearly 40% of patients [31,32]. Several studies have shown that the residual blood flow carried by collateral vessels at the time of AMI exerts some beneficial effects such as reduction in infarct size [33], improvement in residual ejection fraction and other indexes of pump function and prevention of left ventricular aneurysm formation. However, a demonstration of whether collateral circulation improves prognosis after AMI is lacking.

AIM OF THE STUDY

- To compare the risk factors like smoking, diabetes mellitus, hyperlipidemia, obesity and hypertension in young patients with myocardial infarction and patients at or more than 55 years age.
- To assess the angiographic features of coronary artery disease between the two groups.
- To compare the presence of collaterals in patients with acute myocardial infarction between the younger and older age groups.
- To assess the Left ventricular function between the two groups.

REVIEW OF LITERATURE

Heart diseases are occurring in Indians 5 to 10 years earlier than in other populations around the world. The first MI attack occurs in 4.4% of Asian women and 9.7% of men at age less than 40 years, which is 2- to 3.5-fold higher than in the West European population and is third highest of all the regions studied worldwide. These studies carried out in India and other places suggest that Asians in general and Indians in particular are at increased risk of MI at a younger age (<40 years), irrespective of whether they have migrated to other countries or are resident Asians.

In South Asians, apolipoprotein (Apo)B/ApoA1 and smoking were the important risk factors, as in the rest of the world. However, hypertension, abdominal obesity, and diabetes had more severe effects in South Asia. The INTERHEART study also showed that hypertension and diabetes were more important risk factors in younger Indian women than men. Earlier studies, mostly in Western populations, have also found an association of the above-mentioned risk factors with the development of CAD.

Diabetes:

The worldwide prevalence of type 2 diabetes mellitus (T2DM) is expected to double within the next two decades, with the greatest increase occurring in Asia and the Indian subcontinent, where it will affect >130 million individuals.[34] Indians tend to develop T2DM in conjunction with central obesity, which is in contrast to other ethnic groups who tend to have a generalized obesity. Diabetes is a well established risk factor for cardiovascular disease. Unlike other traditional risk factors, the prevalence of T2DM is found to be uniformly higher in Indians than in other ethnic populations.[35,36]. Even blood glucose concentrations within the higher side of the normal range increases the risk of CVD in Indians.[37] Prevalence of insulin resistance in healthy, young, lean Indian men is three- to fourfold greater than lean men of other ethnic groups. Furthermore, increased prevalence of insulin resistance in Indian men is associated with a twofold increase in hepatic triglyceride and plasma interleukin 6 (IL6) concentrations, when compared with Caucasian men. a twofold increase in hepatic triglyceride and plasma interleukin 6 (IL6) concentrations, when compared with Caucasian men.

India alone is projected to experience the greatest global increase in T2DM by 2025. [38,39] In rural settings, the prevalence of diabetes is quite

low; however, it rises dramatically in urban communities throughout India and even more so among South Asian immigrants to the western world. Significantly higher risk of diabetes mellitus among South Asians including Indians could be due to the so-called "thrifty gene" hypothesis, which suggests an interaction between genetic predisposition and environmental factors. A study by Chiu *et a*[42] has shown that polymorphism of the hepatic glucokinase promoter gene can cause hepatic insulin resistance in Asian Indians. A recent study showed that promoter polymorphism *-482T* and *-455C* of *APOC3* gene is associated with the metabolic syndrome in South Asians.[43] The Ala54Thr polymorphism in the fatty acid binding protein-2 (*FABP2*) gene as well as *T-455C* and *C-482T* polymorphism in apolipoprotein C-III (*APOC3*) gene promoter polymorphism.[44] There are significant ethnic differences in the pathogenesis of insulin resistance between Asian Indian men and Caucasian men, which may have important therapeutic implications for prevention and treatment of T2DM in this group.

Lipid profile:

Hypercholesterolaemia is a well known risk factor for CVD, but the pattern of lipid profile in Indians is very different from other populations. High triglyceride (TGL) concentration, low concentrations

of high density lipoprotein (HDL) and increased visceral fat are more prevalent among Indians. In addition to the actual concentrations of these lipids, particle size appears to be an important predictor of CVD risk. Even though Indians have comparable values of low density lipoprotein (LDL) cholesterol to other populations, the LDL particle size tends to be smaller. The small particle size increases the susceptibility to oxidation, thereby rendering these particles more atherogenic than the larger ones. Similarly, Indians have lower values of HDL with a higher concentration of small HDL particles that are less protective. This type of HDL cholesterol was also observed in Indian men with apparently normal HDL values In Indian women, the concentration of HDL cholesterol was more important than total cholesterol in determining the CVD risk.

Obesity:

The waist-to-hip ratio (WHR) is a measure of abdominal obesity and is a surrogate measure for visceral fat deposition. It was also found that high WHR observed among Indians compared with individuals from other countries contributed to the higher rates of CVD in this population. The diagnostic criteria for the metabolic syndrome by the National Cholesterol Education Program Adult Treatment Panel III (NCEP, ATP III) in the USA has incorporated population specific

definitions for abdominal obesity. Recently, the International Diabetes Federation (IDF) recommended a new definition for metabolic syndrome. This definition included three major modifications as compared to the NCEP, ATP III definition: (1) central obesity has been made a mandatory variable; (2) the cut-off level for waist circumference have been lowered (male 94 cm, female 80 cm), and made ethnicity specific (for example, for South Asians(including Indians) male 90 cm; female 80 cm and (3) the cut-off level for fasting plasma glucose has been lowered to 100 mg/dl (5.5 mmol/l). The severity of insulin resistance increases with increasing adiposity. Body composition of Indians is conducive to development of the metabolic syndrome, as they have a high percentage of body fat, abdominal obesity, insulin resistance, hyperinsulinaemia and lower muscle mass. There has been considerable debate as to whether the underlying cause of the metabolic syndrome is genetically or environmentally determined. Approximately 20–25% of urban Indians have evidence of the metabolic syndrome. Subjects with metabolic syndrome face a twofold increased risk of all cause mortality and a two- to threefold increased risk of cardiovascular mortality. Furthermore, insulin resistance was reported to be present in nearly 30% of children and adolescents in India, more so in girls. Conventional body mass index (BMI) classifications are overweight (25.0 kg/m^2 to <30.0 kg/m^2) and obese (BMI ≥30.0 kg/m^2) and these

cut-off points were derived primarily in European populations to represent the thresholds for the risk of CVD. WHO has therefore revised the classification of obesity in Asians(including Indians) from BMI >30 kg/m^2 to >25 kg/m^2.

Smoking:

Smoking tobacco is an established but modifiable risk factor for CVD. Smoking causes endothelial dysfunction and can also precipitate coronary vasospasm. Risk of CVD in smokers is the same in Indians and other ethnic populations. But the irony is that the prevalence of smoking is increasing in India and south Asia whereas it is decreasing in the developed countries. This could be because of the increased restrictions towards smoking in the western countries, thereby leading the tobacco companies to target the populations in developing countries. The consumption of tobacco either as cigarette or beedi (Indian cigarette made of tobacco wrapped in a leaf and secured with a colored thread at one end) was once low among Indian males and almost unheard of in females, but this is certainly on the rise now. Smoking was one of the most important risk factor for CVD with a dose related relationship in Indians.

Although conventional risk factors account for the majority of CVD risk, the identification of newer methods of risk stratification has

been an area of active research. The role of non-traditional risk factors such as lipoprotein (a) or homocysteine, which are elevated in Indians, in causing CVD is unclear. Among these risk factors, lipoprotein (a), apolipoprotein B, homocysteine, and plasminogen activator inhibitor-1 values tend to be similar in most populations. Recent randomised trials to reduce homocysteine concentrations have not demonstrated a reduction in CVD.

Inflammation plays a central role in the development and progression of atherosclerosis. High C reactive protein (CRP) concentration was independently and positively associated with CVD after adjustment for Framingham risk factors, atherosclerosis, anthropometric measurements, and ethnicity. The mean CRP values were higher in Indians than in Chinese and Europeans, and this effect remained significant even after adjustment for age, sex and metabolic factors. Inflammatory biomarkers like adipokines derived from adipose tissue have been proposed to link insulin resistance to atherosclerosis. Tumour necrosis factor α, interleukin-6, leptin, plasminogen activator inhibitor-1, angiotensinogen, resistin, and CRP are the various adipokines. Insulin resistant states are associated with low adiponectin values, and thus by increasing adiponectin production we would be able to decrease cardiovascular risk. Indians have lower adiponectin

concentrations than Caucasians. The effects of prothrombotic (fibrinogen, plasminogen activator inhibitor-1) or proinflammatory factors (lipoprotein (a), homocysteine) in the causation of CVD needs to be studied. Microalbuminuria, a marker of renal damage, is also an independent risk factor for CVD. High urinary albumin excretion and microalbuminuria were more frequent in Indians.

The prevalence of protective factors like regular physical activity, regular moderated amounts of alcohol intake, and daily intake of fruits and vegetables were notably lower in Indians. Alcohol consumption did not appear to be protective in Indians who have a binge drinking pattern compared to regular controlled drinking that is protective. The higher risk of CVD in Indians has been attributed to ghee (clarified butter) consumption. A high prevalence of ghee consumption has also been reported in migrant Indians living in the west. A recent study showed a positive association between ghee consumption and acute myocardial infarction. In Indian households, prolonged cooking of vegetables is a common practice, which may destroy nearly 90% of the folate content; henceforth the protective effect of the folate is decreased. A similar inverse association between intake of vegetables and CVD has been reported in a case–control study from India.

To the contrary, there is a significant protective effect of vegetarianism and the protective effect was also found among non-vegetarians due to low meat and fish consumption in this population. Consumption of green leafy vegetables and fruits are associated with lower risk of CVD and a gradient towards lower risk is associated with a greater number of servings consumed.

Physical activity increases insulin sensitivity and high density lipoprotein cholesterol, lowers blood pressure, improves endothelial function, and reduces the risk of T2DM, hypertension, and central obesity. A recent study from two Indian cities indicates that daily moderate intensity walking such as brisk walking for 35–40 mins was associated with >50% reduction in risk for CVD. Although physical activity was protective in Indians, the proportion of Indians who regularly exercised was low compared with other regions. For example, leisure time physical activity is culturally unacceptable for most Muslim women. Rural populations in India have higher energy expenditure due to higher work related labour and heavy household duties when compared to the urban population. Adverse psychosocial factors like depression and stress at work or home were found to have a positive association with CVD, consistent with the data from various studies. There are also reports positively correlating a low level of education and

acute myocardial infarction in Indians. This could be due to high risk activities such as smoking, binge drinking, etc, contributing to cardiovascular diseases in individuals with low levels of education.

Fetal origin hypothesis:

There is an increased incidence of intrauterine growth retardation in developing countries. For example, in India alone the mean full term birthweight is <2.7 kg, which is almost 1 kg lower than in the European population. It has been postulated that the low birth weight contributes to CVD and metabolic syndrome in Indians. In Pune Children's Study, CVD risk factors were followed up in 201 children born in a single institution. At the age of 4 years, lower birth weight children had higher plasma insulin and glucose concentrations after an oral glucose load and higher IGF-I concentrations. On further follow up at the age of 8, there was an inverse relationship between birth weight and the incidence of metabolic syndromes. An interesting feature of this study is that the most adverse risk profile was found in children who were small at birth but had a high weight and fat mass at 8 years. All these data suggest that insulin resistance was associated with an increased body mass in children which is further exacerbated by low birth weight.

- Indians have a higher prevalence of cardiovascular disease (CVD).

- High triglyceride concentrations, low concentrations of high density lipoproteins, and increased visceral fat are more prevalent among Indians.

- Central obesity is considered as a more important predictor and risk factor for CVD than generalised obesity.

- The World Health Organization has revised the classification of obesity in South Asians from body mass index >30 kg/m^2 to >25 kg/m^2.

- Not only is the prevalence of diabetes in Indians higher but they also have three- to fourfold higher mortality rates due to CVD than people with diabetes from other ethnic groups.

- Higher levels of risk factors in Indians may be primarily responsible for their higher rates of CVD, therefore aggressive screening for and modification of traditional risk factors in Indians at an early age could substantially reduce the high rates of early onset coronary heart disease in this population.

The phenomenal rise in diabetes and CHD in Indians has been suggested to have its origin in intrauterine life. The thrifty phenotype hypothesis—that is, the "fetal origins" theory—is suggested for the epidemic of CVD and metabolic syndrome due to intrauterine growth

retardation. This hypothesis is based on the observed inverse relationship between birth weight and the risk of diabetes and metabolic syndrome in Indians. Another hypothesis suggesting fetal origin of diabetes is the "fetal insulin" hypothesis, showing an association between low birth weight and diabetes. It has also been proposed that malnutrition during intrauterine life causes irreversible changes in the organ systems of the developing fetus. This increases susceptibility to diseases in later life. Although there is a possibility of multiple insults during development of the fetus, investigators blamed under nutrition as the most likely cause. The possibility that various factors could contribute to the increased risk of CVD and metabolic syndrome in Indians should not be dismissed. The relationships among maternal nutrition, fetal nutrition and later diabetes appear to be more complicated. This may have important implications for developing preventive strategies during pregnancy.

Under nutrition during fetal and early postnatal life may also be associated with development of coronary heart disease, stroke, diabetes, and the metabolic syndrome. These diseases have been shown to have an increased incidence in low birth weight babies. In addition, low socioeconomic status is also related to cardiovascular risk factors due to

physical inactivity, smoking, diabetes, heavy alcohol consumption, high blood pressure, and poor dietary habits.

Coronary collateral circulation:

It is estimated that collateral circulation to infarct-related artery (IRA) is present in almost 40% of patients with acute myocardial infarction (AMI) in the acute phase. Collateral circulations exert beneficial effects by increasing myocardial salvage and preventing ventricular remodeling, thereby improving in-hospital prognosis. Although all aspects of the mechanisms underlying the development of coronary collateral circulation are not well-established, circulation in collateral arteries may be not a simple process of passive dilation of pre-existing collateral channels. Because collateral circulation to an IRA is enhanced with preinfarction angina, it has been suggested that they are not only functionally new arteries originating from a pre-existing arteriole, but also are associated with active proliferation and remodeling by growth of pre-existing arteriolar connections into true collateral arteries. Collateral circulation was graded by using a semiquantitative scale from 0 to 3, depending on the angiographic findings of the occluded artery using the best injection : 0 = no collateral circulation; 1 = collateral filling of side branches without visualization of any epicardial segments; 2 = collateral partially filling the epicardial

segment; 3 = collateral completely filling the epicardial segment. Grades 1 to 3 were defined as the presence of collateral circulation to the IRA.

***Factors contributing to circulation from collaterals*:**

Factors that may control the presence of collaterals after the onset of AMI have been demonstrated in previous clinical studies. The extent of collateral circulation is affected by the presence of multivessel disease, previous MI, preinfarction angina, history of angina pectoris, and time from onset to cardiac catheterization.

Collateral formation is a complex process involving mechanical factors and angiogenic factors, although it is mainly influenced by endothelial function. Endothelial cell function is compromised with increasing age. Therefore, age-dependent endothelial dysfunction may contribute to impaired collateral development in the setting of myocardial ischemia.

In recent years, alternative methods of evaluating coronary collateral circulation have been proposed on the basis that angiography lacks sufficient sensitivity. It is certain that angiography cannot detect collateral vessels ,100 mm in diameter and can miss some intramyocardial collateral vessels. However, it is also certain that all normal human hearts have, among the territories of the major coronary arteries, small anastomotic vessels that range up to 200 mm in diameter. Because collateral vessels that are too small to be visualized

angiographically probably have no clinical impact, we believe that angiography offers sufficient sensitivity for clinical purposes. Several studies have demonstrated that collateral circulation at the time of AMI reduces infarct size, an effect that might explain, at least in part, how collateral vessels protect against cardiogenic shock and mortality When total arterial flow is reduced by coronary stenosis or occlusion, coronary vasodilator reserve is exceeded and flow to the ischemic region is distributed along a gradient that is inversely related to the gradient of intramyocardial pressure. Therefore, the small amounts of blood flow provided through the collateral route are distributed in a nonuniform pattern, being preferentially shunted to the subepicardial zone where intramyocardial pressure is lowest The lower incidence of cardiogenic shock in patients with collateral vessels could be explained, at least in part, by the preservation of an epicardial rim of viable myocardium when operating collateral vessels are present at the time of infarction. This phenomenon could ameliorate the systolic dysfunction and promote a partial functional recovery of the involved wall segment early after AMI. This mechanism could also explain the observation of others of a low incidence of infarct expansion and aneurysm formation in patients with well developed collateral channels at the time of infarction.

Effect of collateral circulation on in-hospital prognosis:

The presence of collateral circulation improves myocardial salvage and prevents ventricular remodeling, thereby improving in-hospital prognosis independent of coronary reperfusion therapy.

MATERIAL AND METHODS

This study was performed in the Department of Cardiology, Government Stanley Hospital, Chennai, during the year 2007 – 2010. The study is a retrospective study involving 142 patients who underwent cardiac catheterization during the period between 2008 and 2010 after myocardial infarction.

STUDY GROUP SELECTION:

Ethical committee clearance was obtained to conduct the study in our hospital.

Inclusion Criteria:

1. Acute ST elevation myocardial infarction
2. Non ST elevation myocardial infarction
3. Both sexes.
4. Age Group <= 40 years and >= 55 years.
5. Both thrombolysed and non thrombolysed patients.

Exclusion Criteria:

1. Age group 41 to 54 years.
2. Chronic stable angina.
3. Unstable angina.

4. Patients who underwent PCI / CABG.
5. Previous MI.

Patient characteristic:

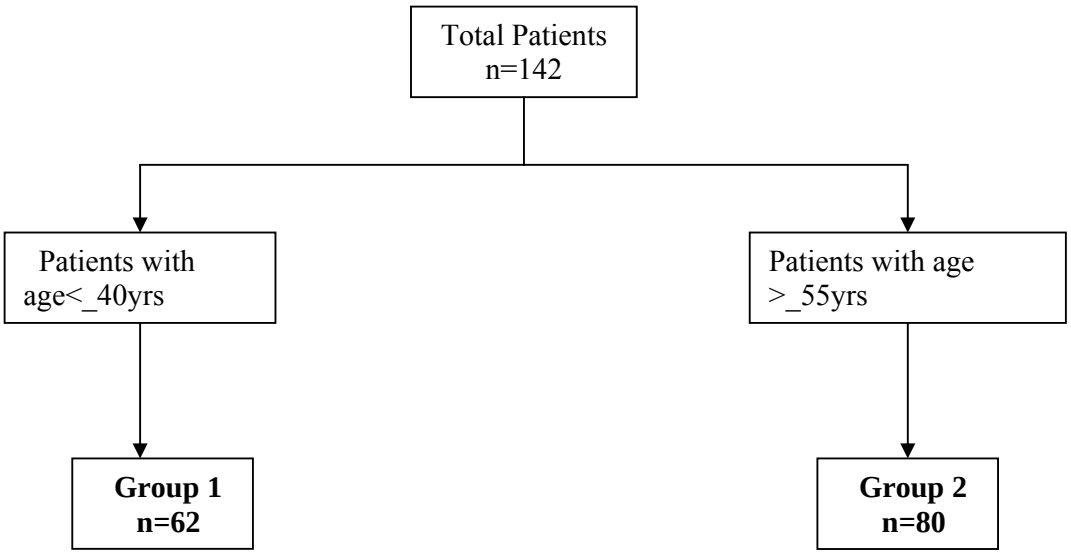
We reviewed all cardiac catheterization and in-hospital records of the first 604 patients who underwent this procedure at Govt. Stanley Medical College, Chennai between 2008 and 2010. Of those 604 patients, 62 patients at or under the age of 40 years has been catheterized for evaluation after myocardial infarction. These 62 young patients were compared to 80 randomly selected patients at or above 55 years of age catheterized for evaluation of coronary artery disease, all whom had suffered a prior myocardial infarction.

The patients were divided into two groups:

Group 1 – Patients less than or equal to 40 years age with myocardial infarction (n = 62).

Group 2 – Patients with 55 years or more than that with myocardial infarction (n = 80). The cut off age of less than or equal to 40 years for young MI group was assigned in accordance with various previous studies which use similar cut off. 45

These 142 patients underwent clinical evaluation, investigations including EKG, cardiac enzymes, lipid profile, body mass index calculation, fasting sugar, Echocardiography and coronary angiography which were reviewed from their in-patient hospital records.



Detailed history was obtained from all the patients, including the presence of risk factors like

-diabetes mellitus,

-hypertension,

-smoking and

-family history of ischemic heart disease.

Baseline investigations were done in all patients including complete blood count, blood sugar, renal function tests, lipid profile, chest X-ray. Cardiac enzymes, namely, transaminase levels, Creatinine kinase and CK-MB were done in all patients. Body mass index were calculated already for all patients as per records.

	GROUP 1(n=62)	GROUP 2(n=80)	TOTAL
AGE	**<_ 40yrs**	**>_55yrs**	
MALE	**54(87.1%)**	**71(88.8%)**	**125(88%)**
FEMALE	**8(12.9%)**	**9(11.3%)**	**17(12%)**
SMOKING	**48(77.4%)**	**50(62.5%)**	**98(69%)**
F/H OF CAD	**11(17.7%)**	**11(13.8%)**	**22(15.5%)**
HYPERLIPIDEMIA	**24(38.7%)**	**64(80%)**	**88(62%)**
DIABETES	**16(25.8%)**	**32(40%)**	**48(33.8%)**
HYPERTENSION	**15(24.2%)**	**39(48.8%)**	**54(38%)**
OBESITY	**3(4.8%)**	**6(7.5%)**	**9(6.3%)**

Diabetes mellitus was defined as a fasting plasma glucose concentration ≥126 mg/dl or the use of ant diabetic therapy. Hypertension was defined as a history of a systolic blood pressure ≥140 mm Hg, a diastolic blood pressure ≥90 mm Hg, or the use of antihypertensive therapy. Hyperlipidemia was defined as a fasting total cholesterol concentration ≥220 mg/dl, a fasting triglyceride concentration ≥150 mg/dl, or the use of antihyperlipidemic therapy. Smoking defined as more than 15 pack-years.

Ethnicity specific values for waist circumference (according to the International Diabetes Federation) was used in our study population which is for male ≥90 cm; female ≥80 cm; Family history of premature coronary artery disease is defined as <55 years male or <65 years female affected with CAD in first degree relatives

Myocardial infarction was diagnosed if all of three of the following criteria were present.1.Sustained chest pain or discomfort typical of cardiac ischemia, lasting longer than 30 minutes and not relieved by nitroglycerin;2.Initial ST elevation or depression of 1mm or more with(Q wave) or without(non-Q wave) in at least two consecutive inferior or more than 2mm ST elevation in at least two consecutive anterior electrocardiographic leads; and 3 elevation of total creatine kinase values , in addition to elevation of the MB creatine kinase fraction values.

All patients had been subjected to lipid profile within 24 hours of admission and their height, weight and body mass index were calculated using the formula.

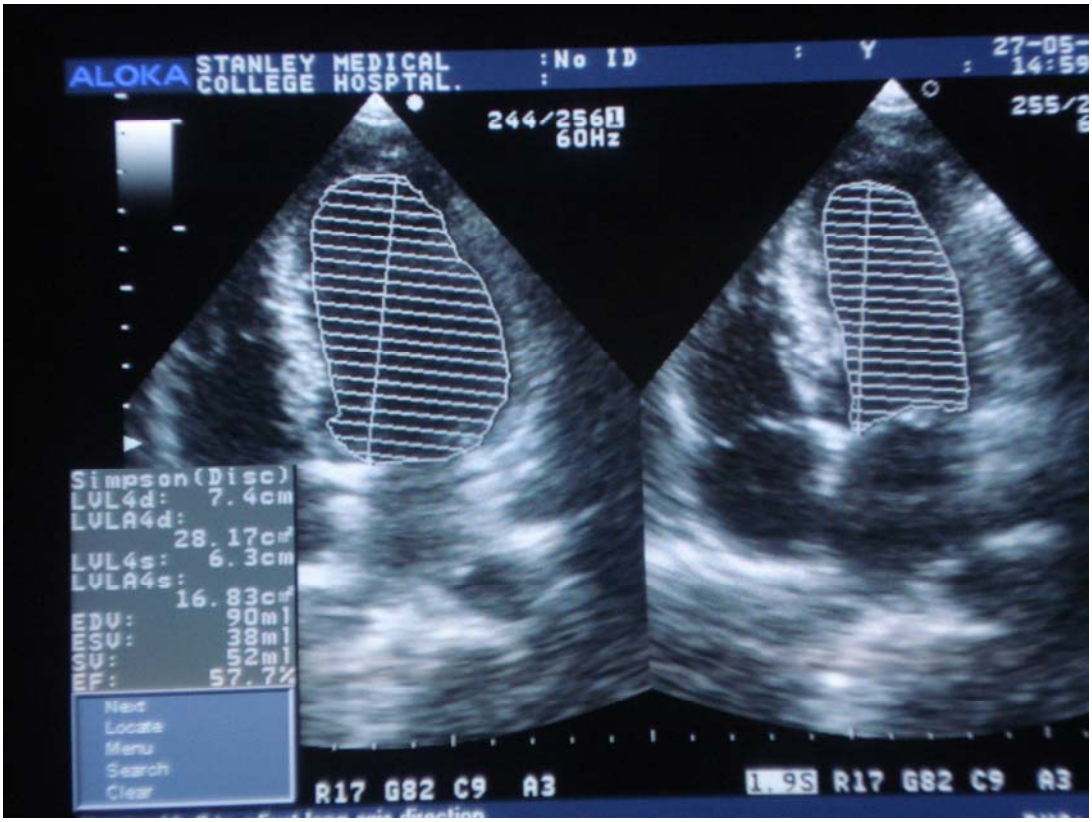
BMI = (Weight in Kilograms / (Height in Meters ^{2})

Echocardiography was done in all patients and ejection fraction and LV function assesessed by modified simpson method.

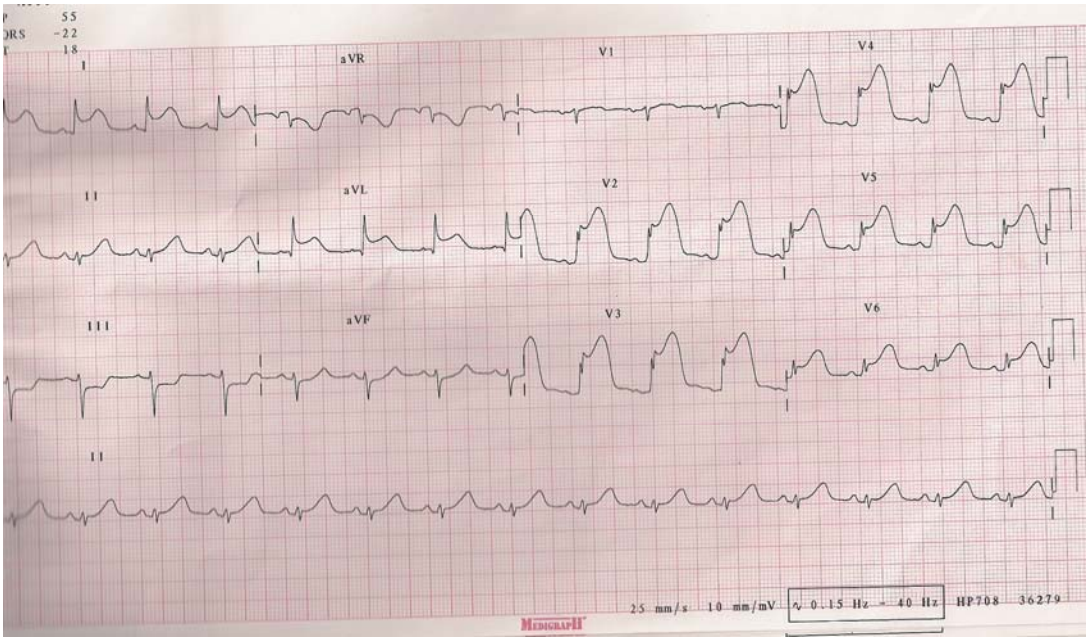
All patients underwent catheterization within two weeks of their hospitalization. The coronary angiography was performed via the femoral artery route using standard sized sheaths and Jud kin's left and right coronary catheters, Amplatz if necessary or via right radial approach using 5 TIG catheters. Multiple angulations and views were used. Lumen diameter narrowing was graded as 0, 25, 50, 75, 90 and 100%. The definition of a significant anatomical stenosis was >70 % localized luminal narrowing.

Percentage of diseased group Vs Age Group			Disease group				Total
			Normal Vessel	One Vessel	Two Vessel	Three Vessel	
Age Group	<= 40	Count	22	21	16	3	62
		% within Age Group	35.5%	33.9%	25.8%	4.8%	100.0%
	>= 55	Count	8	19	23	30	80
		% within Age Group	10.0%	23.8%	28.8%	37.5%	100.0%
Total		Count	30	40	39	33	142
		% within Age Group	21.1%	28.2%	27.5%	23.2%	100.0%

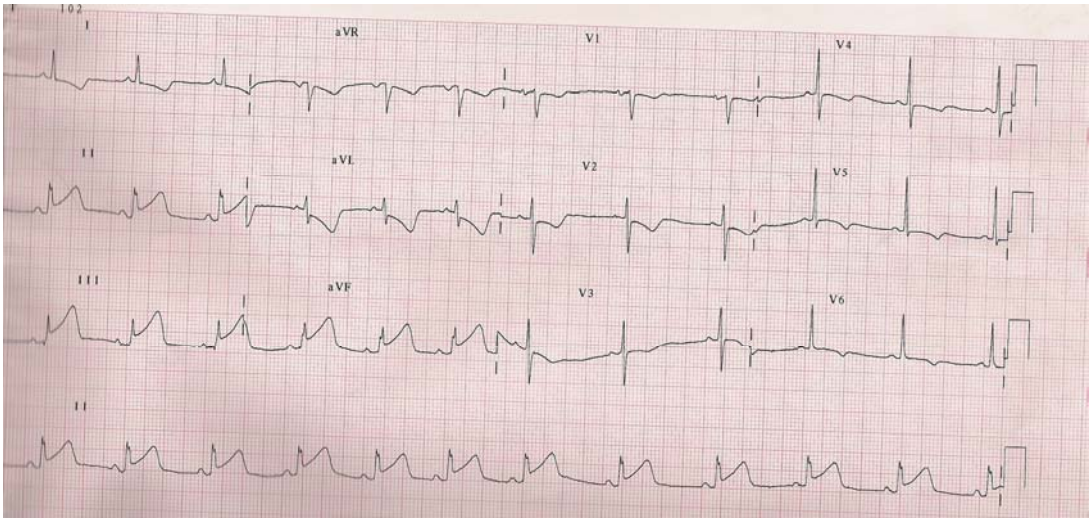
We analyzed the collateral circulation. It was assessed in coronary cine angiograms by two experienced coronary angiographers who had no knowledge of patient data.. Collateral circulation was graded by using a semi quantitative scale from 0 to 3. 0 = no collateral circulation; 1 = collateral filling of side branches without visualization of any epicardial segments; 2 = collateral partially filling the epicardial segment; 3 = collateral completely filling the epicardial segment. Grades 1 to 3 were defined as the presence of collateral circulation to the IRA.



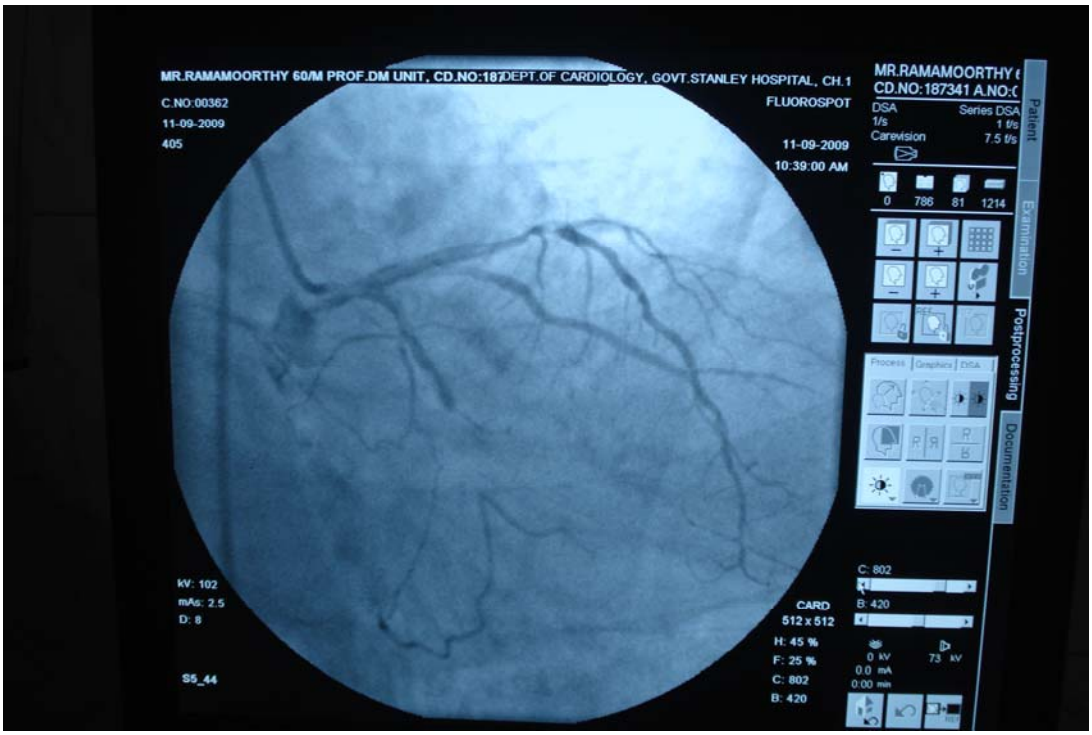
Modified Simpson method



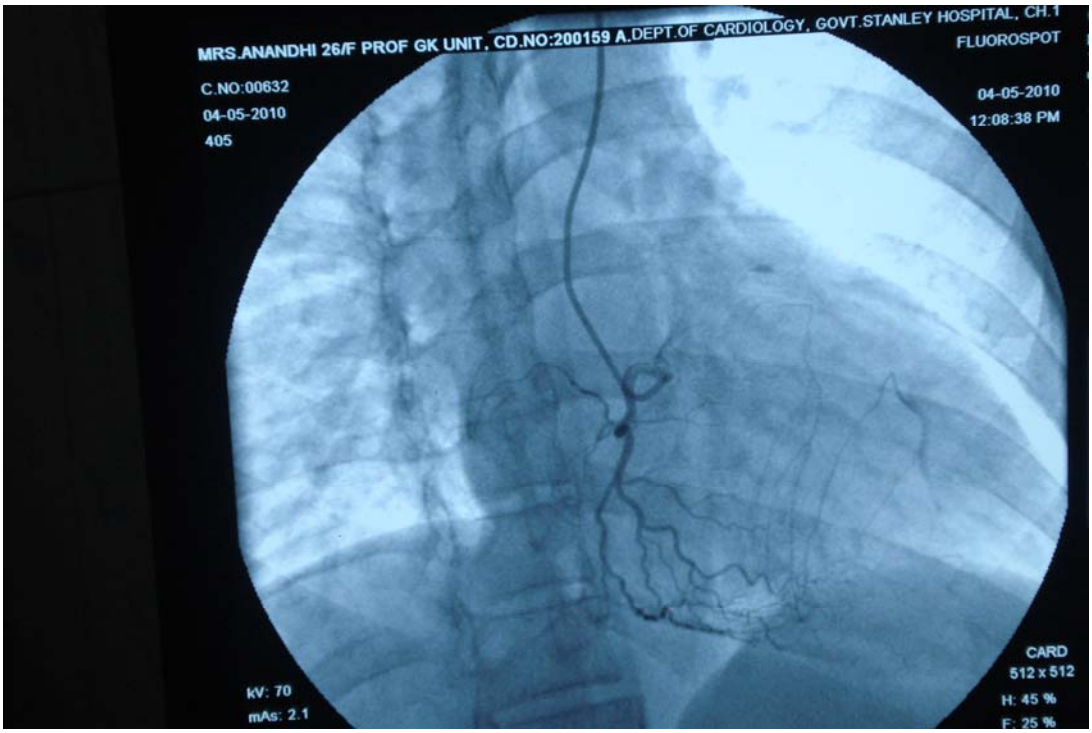
Acute extensive anterior wall myocardial infarction



Acute inferior wall myocardial infarction

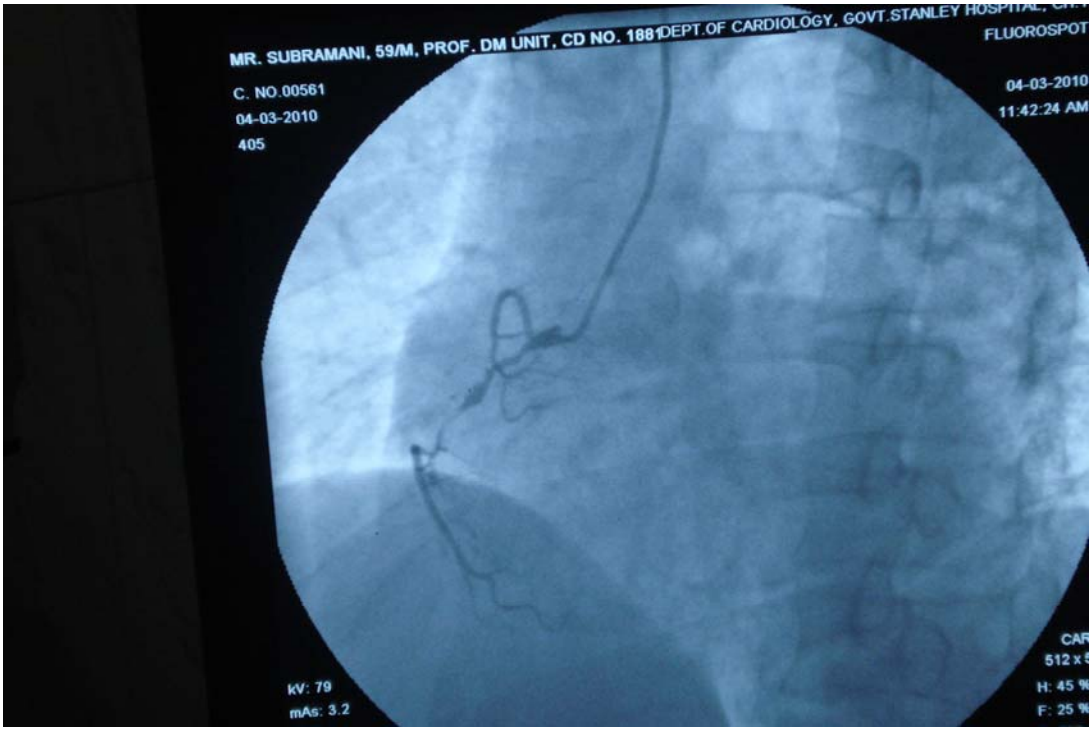


Homo collaterals from proximal to distal LCX.



Septal collaterals from right PDA to LAD

Collaterals from left system to right PDA



Proximal and Mid RCA lesion.

RESULTS

During the study period, 142 patients were evaluated who underwent cardiac catheterization and echo evaluation.. Of the 62 patients aged 40 or less (group 1) 54 were men and 8 were women. In group2 (more than or equal to 55 years) 71 were men and 9 were women. The sex distribution was not significantly different between two groups.

Table 1 Sex

			Sex		
			Male	Female	Total
Age Group	<= 40	Count	54	8	62
		% within Age Group	87.1%	12.9%	100.0%
	>= 55	Count	71	9	80
		% within Age Group	88.8%	11.3%	100.0%
Total		Count	125	17	142
		% within Age Group	88.0%	12.0%	100.0%

Chi-square value = 0.091, p = 0.763 (Non significant).

Risk factor analysis revealed smoking to be overwhelmingly present in both groups (77.4 percent [48] in group 1 and 62.5 percent [50] in group 2).

There is no statistically significant difference between group 1 and group2 with respect to smoking as a risk factor for CAD.

Table – 2 Smoking

			Smoking		
			No	Yes	Total
Age Group	<= 40	Count	14	48	62
		% within Age Group	22.6%	77.4%	100.0%
	>= 55	Count	30	50	80
		% within Age Group	37.5%	62.5%	100.0%
Total		Count	44	98	142
		% within Age Group	31.0%	69.0%	100.0%

Chi-square Value=3.636, p=.057 (Non significant)

When compared in group1 family history was not statistically significant (p value = 0.514) with group2. Although young MI group (group 1) had more frequent family history than group2.

Table 3 Family History

			FH		
			No	Yes	Total
Age Group	<= 40	Count	51	11	62
		% within Age Group	82.3%	17.7%	100.0%
	>= 55	Count	69	11	80
		% within Age Group	86.3%	13.8%	100.0%
Total		Count	120	22	142
		% within Age Group	84.5%	15.5%	100.0%

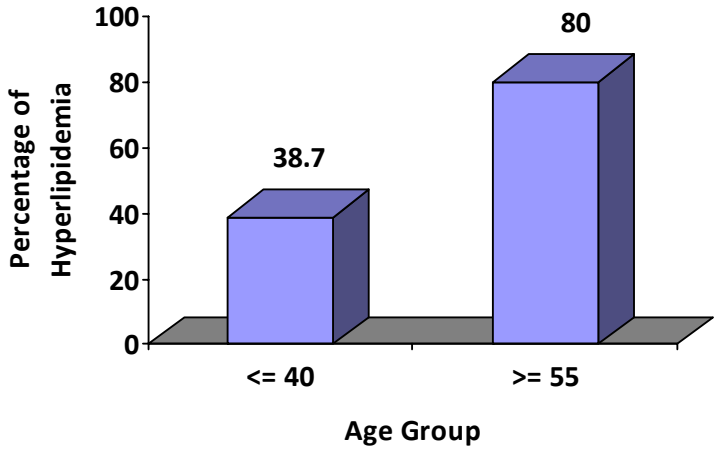
Chi-square Value=.425, p=0.514 (Non significant)

Hyperlipidemia was more common in group2 64 [80%] patients while compared with 24 [38.7 %] in group1 patients were more common in group 1 but were not statistically more frequent. p value was significant ($p < 0.001$) .

Table 4 - Hyperlipidemia

			Hyperlipidemia		
			No	Yes	Total
Age Group	<= 40	Count	38	24	62
		% within Age Group	61.3%	38.7%	100.0%
	>= 55	Count	16	64	80
		% within Age Group	20.0%	80.0%	100.0%
Total		Count	54	88	142
		% within Age Group	38.0%	62.0%	100.0%

Chi-square Value=25.269, p<0.001 (Significant)



In group1 there were 16 patients [25.8%] with diabetes when compared with group2 which had 32 patients [40%] . Though there is no

statistical significance between two groups (p = 0.076 – non significant) there were more diabetic in group 2.

Table 5 – Diabetes Mellitus

			DM		
			No	Yes	Total
Age Group	<= 40	Count	46	16	62
		% within Age Group	74.2%	25.8%	100.0%
	>= 55	Count	48	32	80
		% within Age Group	60.0%	40.0%	100.0%
Total		Count	94	48	142
		% within Age Group	66.2%	33.8%	100.0%

Chi-square Value=3.145, p=.076 (Non significant)

In our study only 9 patients [6.3%] were obese. There was no significant difference between the two groups with regard to obesity.

Table 6 - Obesity

			Obesity		
			No	Yes	Total
Age Group	<= 40	Count	59	3	62
		% within Age Group	95.2%	4.8%	100.0%
	>= 55	Count	74	6	80
		% within Age Group	92.5%	7.5%	100.0%
Total		Count	133	9	142
		% within Age Group	93.7%	6.3%	100.0%

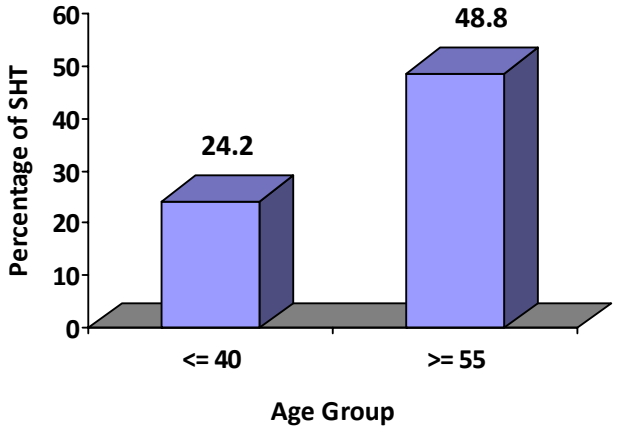
Chi-square Value=.417, p=.519 (Non Significant)

Hypertension was more frequent in group 2 patients [48.8 %] than in group1[24.2%]. There is a statistical significant p value (p=0.003) between two groups.

Table 7 – Hypertension

			SHT		
			No	Yes	Total
Age Group	<= 40	Count	47	15	62
		% within Age Group	75.8%	24.2%	100.0%
	>= 55	Count	41	39	80
		% within Age Group	51.3%	48.8%	100.0%
Total		Count	88	54	142
		% within Age Group	62.0%	38.0%	100.0%

Chi-square Value=8.938, p=0.003 (Significant)



There were more risk factors per patient in group2 (more than 3 risk factors = 52.5%) when compared with group1 (17.7%) .

Table 8 Risk factor

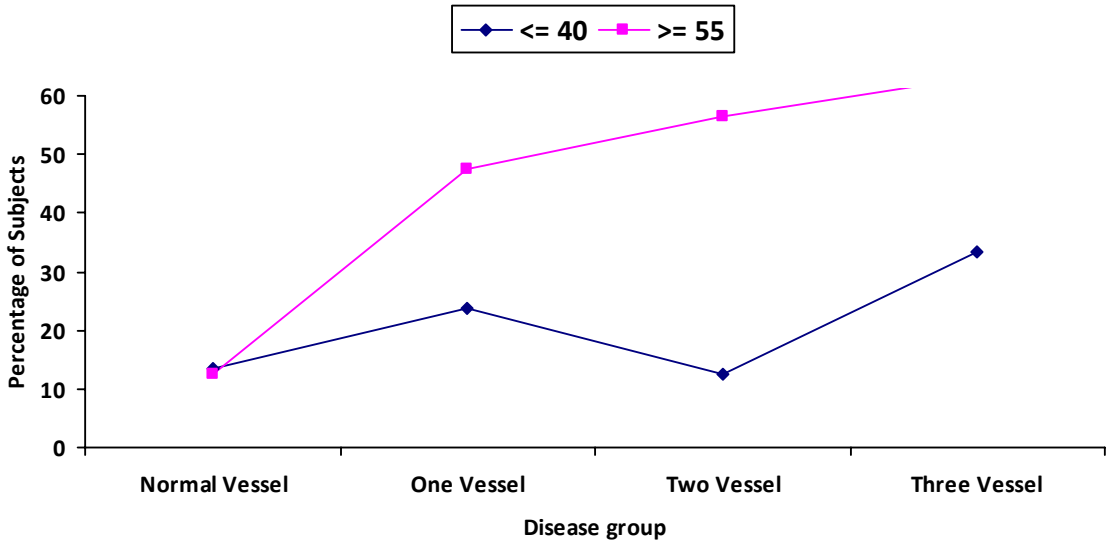
Risk factor --<3 Risk factor Vs >=3 Risk factor for both the age group

Age Group * Risk_Fact_grp Crosstabulation

			Risk_Fact_grp		
			<3 RF	>= 3 RF	Total
Age Group	<= 40	Count	51	11	62
		% within Age Group	82.3%	17.7%	100.0%
	>= 55	Count	38	42	80
		% within Age Group	47.5%	52.5%	100.0%
Total		Count	89	53	142
		% within Age Group	62.7%	37.3%	100.0%

Chi-square Value=18.04, p<0.001 (Significant)

Subjects with >=3 risk Factors in various diseases and age group

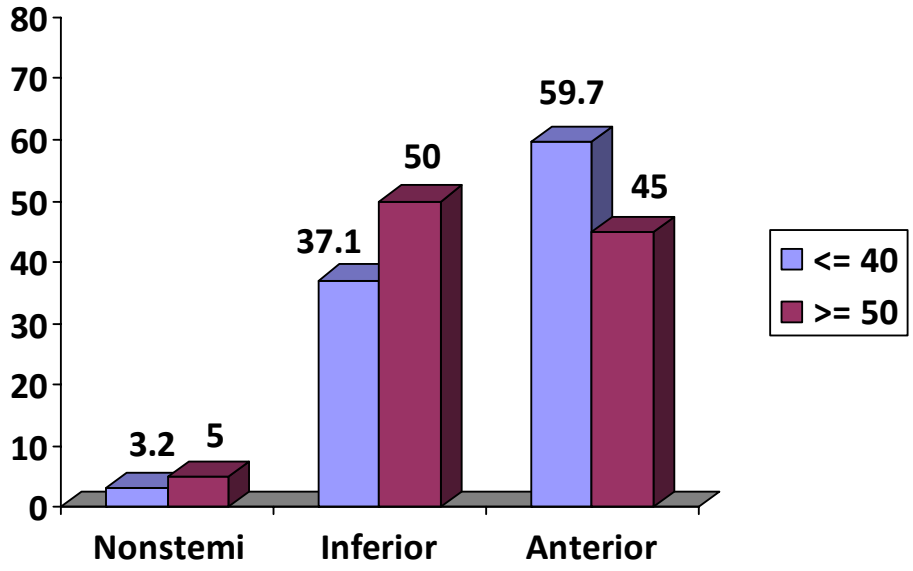


Age Group <=40 - Chi-square Value=.135, p<0.713 (Non Significant)
Age Group >=55 - Chi-square Value=5.54, p<0.019 (Significant)

In our study, group1 patients had suffered 59.7 percent [37] anterior wall myocardial infarction(MI) , 37.1 percent [23] inferior MI while group2 patients had 45 percent [36] and 50 percent [40] ,anterior and inferior wall MI respectively. Small number of patients had Non ST elevation myocardial infarction (NSTEMI) in both groups.

Table 9 Myocardial Infarction

			MI			
			Nonstemi	Inferior	Anterior	Total
Age Group	<= 40	Count	2	23	37	62
		% within Age Group	3.2%	37.1%	59.7%	100.0%
	>= 55	Count	4	40	36	80
		% within Age Group	5.0%	50.0%	45.0%	100.0%
Total		Count	6	63	73	142
		% within Age Group	4.2%	44.4%	51.4%	100.0%



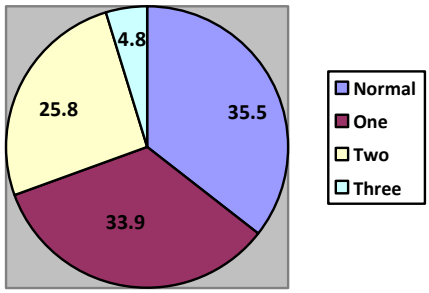
Chi-square Value=3.035, p=0.219 (Non significant)

Angiographically, the incidence of one vessel disease and normal coronary anatomy was much higher in patients from group 1. In group 2 there were 10% with normal coronary anatomy and 23.8% with one vessel disease involvement. There was almost an equal prevalence of two-vessel disease, between the two groups. Group 1 being 25.5% and 28.8% respectively. But the incidence of significant three-vessel disease was statistically much lower in group 1 vs. group 2, with incidences of 4.8 % [3] and 37.5% [30], respectively .

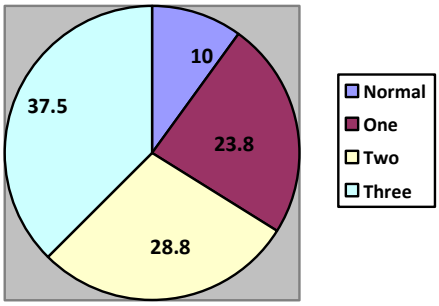
Table 10 Percentage of diseased group Vs Age Group

			Disease group				
			Normal Vessel	One Vessel	Two Vessel	Three Vessel	Total
Age Group	<= 40	Count	22	21	16	3	62
		% within Age Group	35.5%	33.9%	25.8%	4.8%	100.0%
	>= 55	Count	8	19	23	30	80
		% within Age Group	10.0%	23.8%	28.8%	37.5%	100.0%
Total		Count	30	40	39	33	142
		% within Age Group	21.1%	28.2%	27.5%	23.2%	100.0%

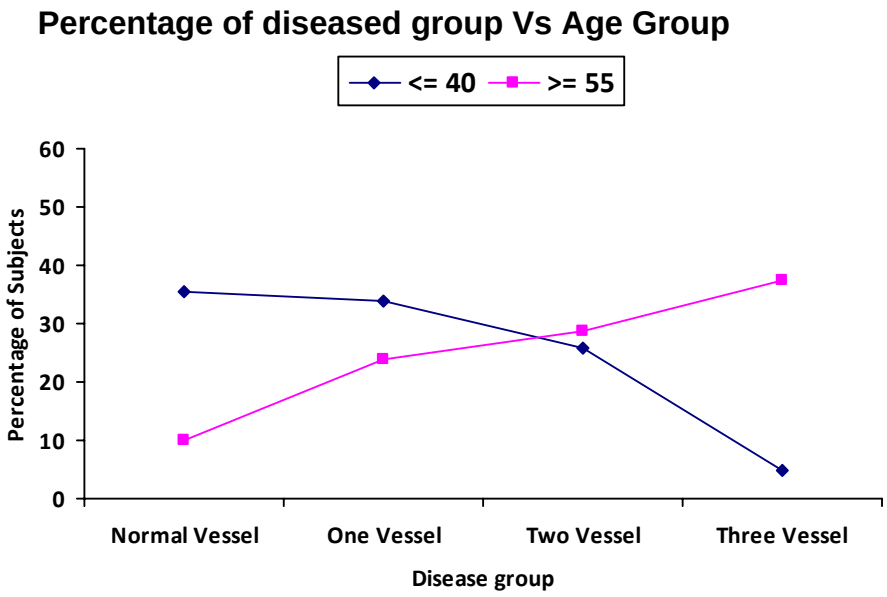
Trend Chi-square Value=26.822, $p<0.001$ (Significant)



Group 1



Group 2



The incidence of left main coronary artery disease was low in group1 6.5percent [4] when compared to group2 which is 17.5 percent[14] which was statistically significant.

Table -11 LMCA

			LMCA		
			No	Yes	Total
Age Group	<= 40	Count	58	4	62
		% within Age Group	93.5%	6.5%	100.0%
	>= 55	Count	66	14	80
		% within Age Group	82.5%	17.5%	100.0%
Total		Count	124	18	142
		% within Age Group	87.3%	12.7%	100.0%

Chi-square Value=3.852, p=0.050 (Significant)

In group 1 there were 51.6 percent [32] of Left Anterior Descending Artery involvement when compared to 70.1 percent [56] in group2. The incidence of proximal LAD involvement was almost double in the group2 patients.

Table 12 LADS

			LAD			
			Normal	Mid	Proximal	Total
Age Group	<= 40	Count	30	19	13	62
		% within Age Group	48.4%	30.6%	21.0%	100.0%
	>= 55	Count	24	23	33	80
		% within Age Group	30.0%	28.8%	41.3%	100.0%
Total		Count	54	42	46	142
		% within Age Group	38.0%	29.6%	32.4%	100.0%

Trend Chi-square Value=6.825, p=0.009 (Significant)

Left circumflex Artery was more involved in group2 patients 56.3 percent [45] than in group1 patients 12.9 percent [8] .

Table 13 LCX – left circumflex

			LCX		
			No	Yes	Total
Age Group	<= 40	Count	54	8	62
		% within Age Group	87.1%	12.9%	100.0%
	>= 55	Count	35	45	80
		% within Age Group	43.8%	56.3%	100.0%
Total		Count	89	53	142
		% within Age Group	62.7%	37.3%	100.0%

Chi-square Value=28.055, p<0.001 (Significant)

The incidence of Right Coronary Artery involvement in group1 is 29 percent [18] and group2 is 65 percent [52] respectively. RCA is more involved in group2 than in group1 with statistical significance.

Table 14 RCA

			RCA		
			No	Yes	Total
Age Group	<= 40	Count	44	18	62
		% within Age Group	71.0%	29.0%	100.0%
	>= 55	Count	28	52	80
		% within Age Group	35.0%	65.0%	100.0%
Total		Count	72	70	142
		% within Age Group	50.7%	49.3%	100.0%

Chi-square Value=18.079, p<0.001 (Significant)

In group1 patients incidence of collaterals was 6.5 percent [4] when compared to group2 patients 26.3 percent [21] respectively. P value (p = 0.002) is significant.

Table 15 Collaterals

			Collaterals		
			No	Yes	Total
Age Group	<= 40	Count	58	4	62
		% within Age Group	93.5%	6.5%	100.0%
	>= 55	Count	59	21	80
		% within Age Group	73.8%	26.3%	100.0%
Total		Count	117	25	142
		% within Age Group	82.4%	17.6%	100.0%

Chi-square Value=9.439, p=0.002 (Significant)

The mean ejection fraction was 51.47 % in group1 and 52.59% in group2.The T-test value was 1.129 and there was no statistical significance between the two groups with respect to the ejection fraction

Table 16 Ejection fraction

	Age Group	N	Mean	Std. Deviation	Std. Error Mean
EF	<= 40	62	51.47	5.734	.728
	>= 55	80	52.59	5.955	.666

T-Test Value=-1.129, p-Value=0.258 (Non Significance)

EF Vs Collaterals

	Collaterals	N	Mean	Std. Deviation	Std. Error Mean
EF	No	117	52.51	5.597	.517
	Yes	25	50.16	6.780	1.356

According to Levene's Test for equality of variances, the sig (2 tailed) is 0.115 which is not statistically significant. There was no difference in ejection fraction between those with and without collaterals.

DISCUSSION

The distribution of lesions in our patients, with a high incidence of normal coronary arteries and one vessel disease, is in accordance with previous studies.[46,47]. In our study patients with young MI (less than or equal to 40 years age) had 35.5%, and patients with age more than or equal to 55 years had 10% normal coronary arteries.

Prospective studies (25-30) of patients -<65 years old who underwent coronary arteriography after myocardial infarction havc found angiographically normal coronary arteries in 0% to 4%. By comparison, prospective and retrospective analyses (3-9) of patients <35 years old have described angiographically normal coronary arteries in 9% to 17%.In the study by, Tewari S et al in Indian Heart Journal 2005 Jul – Aug, one vessel involvement more common in patients < or = 40 years age group. The incidence of two vessel disease was similar between the less than or equal to 40 years and more than or equal to 55 years in our study. M.W Wolfe and J L Vacek et al in Chest 1988, in their studies had shown that the incidence of two vessel was similar between the two groups as if in our study. The actual prevalence of angiographically normal coronary arteries with myocardial infarction has been difficult to determine because some reports (5,8,9) have

included patients with up to 50% coronary stenosis as well as those free of lumen narrowing. Others (3, 6, 10, 25, and 28) have distinguished patients with moderate disease from those with angiographically normal arteries. Biswas PK et al in Journal of Indian Medical Association 1995 had documented the preponderance of one vessel disease in young MI patients.

Young patients with significant coronary obstruction have less extensive disease than older patients. The present findings of predominantly single-vessel disease in young patients and multivessel disease in older patients is in accord with previous studies [48]. In our study, the left main coronary artery disease is higher (17. 5 %) in patients with age more than or equal to 55 years than in young MI patients. In younger age group (less than or equal to 40 years age group), there were 51.6 percent [32] of Left Anterior Descending Artery involvement when compared to 70.1 percent [56] in more than or equal to 55 years group.

The incidence of proximal LAD involvement was almost double in the more than or equal to 55 years patients.

Patients in more than or equal to 55 years age group [26.3%] had more collaterals than young MI group[6.5%] which was statistically significant.Toshiya Kurotobi et al in Journal of American Coll Cardiology 2004, has noted that ,time to catheterization, history of angina pectoris, and preinfarction angina were independent predictors for the presence of collaterals.In our study , Patients in elderly age group had more frequent pre infarction angina than in younger group. [55]

The mean ejection fraction was 51.47 % in less than or equal to 40 years and 52.59% in more than or equal to 55 years. The T-test value was 1.129 and there was no statistical significance between the two groups with respect to the ejection fraction. There was no statistically significant difference in ejection fraction between patients with and without collaterals.

There is no statistically significant difference between younger age group and more than or equal to 55 years with respect to smoking as a risk factor for CAD. In our study, smoking was overwhelmingly present in both groups than any other risk factors. The incidence of 77.4% smoking in the younger age group in our study correlates with previously reported data which ranges from 80 – 86 %. [49-50]

When compared with younger age (less than or equal to 40 years) family history was not statistically significant (p value = 0.514) in more than or equal to 55 years group. Although young MI group (group 1) had more frequent family history than group2. This is in correlation with the previous studies showing family history of premature coronary heart disease was more common only in young men and reported to be greater [51,52] or no different [53,54] according to age.

Franklin H Zimmerman et al in CASS registry has found the levels of Cholesterol and triglycerides to be similar in all groups. But, in our study hyperlipidemia was more common in more than or equal to 55 years age group 64 [80%] patients while compared with 24 [38.7 %] patients in younger age group. p value was significant ($p < 0.001$).

In young MI group there were 16 patients [25.8%] with diabetes when compared with more than or equal to 55 years which had 32 patients [40%]. Though there is no statistical significance between two groups ($p = 0.076$ – non significant) there were more diabetics in group 2.

In our study only 9 patients [6.3%] were obese. There was no significant difference between the two groups with regard to obesity.

Hypertension was more frequent in group 2 patients [48.8 %] than in less than or equal to 40 years [24.2%]. There is a statistical significant p value ($p=0.003$) between two groups.

Wolfe MW, Vacek JL. et al in Myocardial infarction in the young. Chest 1988, hypertension and diabetes were more common in the elderly age group than in younger group. But in our study hypertension and hyperlipidemia were more common in elderly group than younger group. Diabetes even though not statistically significant was still more frequent in elderly group.

There were more risk factors per patient in more than or equal to 55 years (more than 3 risk factors = 52.5%) when compared with less than or equal to 40 years (17.7%).

LIMITATIONS

Our study did not include all patients with a myocardial infarction to routinely undergo cardiac catheterization. Hence, the present analysis cannot claim to represent the findings for all patients early after myocardial infarction. Coronary angiography was performed according to clinical indication for suspected coronary artery disease and was representative of clinical practice. Therefore, there is a potential for selection bias.

No attempt was made to document the presence of coronary vasospasm in the patients with normal coronary arteries by ergonovine stimulation, due to the patients being catheterized during the period early after infarction. No systemic questioning regarding the use of cocaine or other sympathomimetic drugs was performed.

CONCLUSION

Young MI patients (age 40 years) patients who have had a myocardial infarction have less extensive coronary artery disease than older (more than or equal to 55 years) patients, with a significant incidence of angiographically normal vessels and with left main.

The risk factor analysis reveals that hypertension and hyperlipidemia were more common in older patients while smoking was common in both the groups being more frequent in younger age group.

To slow the momentum of Coronary artery disease , particularly among the working-age population, major initiatives are needed to combat CAD, whether promotion of diet and physical activity, generation of awareness among both sexes, or development of guidelines for risk factors and therapeutic and surgical strategies.

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ANNEXURE I

ABBREVIATIONS

AMI	-	Acute Myocardial Infarction
CAD	-	Coronary Artery Disease
CVD	-	Cardio Vascular Disease
EF	-	Ejection Fraction
HDL	-	High Density Lipoprotein
LAD	-	Left Anterior Descending Artery
LCX	-	Left Circumflex Artery
LDL	-	Low Density Lipoprotein
LMCA	-	Left Main Coronary Artery
LVH	-	Left Ventricular Hypertrophy
NONSTEMI	-	NON ST Elevation Myocardial infarction
PAR	-	Population Attributable Risk
RCA	-	Right Coronary Artery
STEMI	-	ST Elevation Myocardial Infarction
T2DM	-	Type 2 Diabetes Mellitus
VLDL	-	Very Low Density Lipoprotein

MASTER CHART 1 - YOUNGER AGE GROUP(<= 40 YEARS)

S.No	Name	Age	Sex	Smoking	MI	F/H	Hyperl	DM	SHT	Obesity	EF %	Disease group	LAD	LCX	RCA	LMCA	Collater
1	Jeyaraman	38	M	Y	Anterior	N	N	N	N	N	47	Normal vessel	N	N	N	N	N
2	Venkatesan	22	M	N	Anterior	N	Y	N	N	Y	56	Normal vessel	N	N	N	N	N
3	Ismail	38	M	N	Anterior	N	N	N	Y	N	50	One vessel	Mid	N	N	N	N
4	Arul	38	M	Y	Anterior	N	N	Y	N	N	53	Normal vessel	N	N	N	N	N
5	Vadivel	40	M	Y	Anterior	N	N	N	N	N	50	Normal vessel	N	N	N	N	N
6	Murugeswari	35	F	N	inferior	N	Y	Y	N	N	60	One vessel	N	N	Y	N	N
7	Srinivasan	32	M	Y	Inferior	N	N	N	N	N	58	One vessel	N	N	Y	N	N
8	Abdulkarim	38	M	Y	Anterior	N	N	N	N	N	52	Normal vessel	N	N	N	N	N
9	Sriram	40	M	Y	inferior	Y	N	N	N	N	50	One vessel	N	N	Y	N	N
10	Abdul Ajeez	40	M	Y	Anterior	N	Y	N	N	N	35	three vessel	Proximal	Y	Y	N	Y
11	Francis	40	M	Y	Anterior	N	Y	Y	Y	N	48	One vessel	Mid	N	N	N	N
12	Narayanan	40	M	Y	Inferior	N	Y	N	N	N	52	three vessel	Proximal	Y	Y	N	N
13	Devi	36	F	N	inferior	N	N	N	Y	N	65	Normal vessel	N	N	N	N	N
14	Ramamoorthy	40	M	Y	Anterior	N	N	N	N	N	48	Two vessel	Proximal	Y	N	N	N
15	RajaKumari	38	F	N	inferior	N	N	Y	Y	N	60	Normal vessel	N	N	N	N	N
16	Thirupal	31	M	Y	Anterior	N	Y	Y	N	N	42	One vessel	Mid	N	N	N	N
17	Kumar	36	M	Y	inferior	Y	N	N	N	N	52	One vessel	N	N	Y	N	N
18	Elumalai	39	M	Y	Anterior	N	N	N	N	N	46	Two vessel	Distal	N	Y	N	N
19	Amirthalingam	35	M	Y	Inferior	N	N	N	N	N	55	Two vessel	Proximal	N	Y	N	N
20	Sundaraj	38	M	N	Anterior	Y	Y	N	N	N	48	One vessel	Mid	N	N	N	N
21	Nivaskar	28	M	N	Anterior	Y	N	N	Y	Y	52	Normal vessel	N	N	N	N	N
22	Saleem	39	m	Y	inferior	Y	N	N	N	N	52	Two vessel	Proximal	N	Y	Y	N
23	Sureshbabu	34	M	Y	Anterior	N	N	N	N	N	62	One vessel	Proximal	N	N	N	N
24	Nijam	41	M	Y	inferior	N	Y	Y	N	N	50	three vessel	Mid	Y	Y	Y	N
25	Tamilmalai	40	M	Y	Anterior	N	Y	N	N	N	40	Two vessel	Proximal	N	Y	N	N
26	Anandhi	26	F	N	Anterior	N	Y	N	Y	N	60	Two vessel	Proximal	Y	N	N	Y
27	Moorthy	40	M	Y	Anterior	N	Y	Y	Y	N	50	One vessel	Mid	N	N	N	N
28	Sheiq mohideen	39	M	Y	Anterior	N	Y	N	N	N	60	Two vessel	Mid	Y	N	N	Y
29	Saravanan	40	M	Y	Anterior	N	Y	N	Y	N	45	One vessel	Mid	N	N	N	N
30	TamilSelvi	40	F	N	Anterior	Y	N	Y	N	N	49	Two vessel	Proximal	N	Y	N	N
31	VijayaRagavan	35	M	Y	Anterior	N	N	N	Y	N	55	One vessel	Mid	N	N	N	N

32	Mohamed Hussain	40	M	Y	inferior	N	N	Y	N	N	50	Two vessel	Mid	N	Y	N	N
33	Jeyapal	38	M	Y	inferior	N	N	N	N	N	52	Normal vessel	N	N	N	N	N
34	Sajiv	25	M	Y	Inferior	N	N	N	N	N	55	Normal vessel	N	N	N	N	N
35	Anandan	36	M	Y	Anterior	N	Y	N	N	N	45	Normal vessel	N	N	N	N	N
36	Prabu	26	M	Y	inferior	N	N	N	N	Y	47	Normal vessel	N	N	N	N	N
37	Sakthivel	39	M	Y	Anterior	N	Y	N	N	N	45	One vessel	Mid	N	N	N	N
38	Nazeer	38	M	Y	Nonstemi	N	N	N	N	N	56	One vessel	Proximal	N	N	N	N
39	Sekar	40	M	Y	inferior	N	N	N	Y	N	60	One vessel	N	N	Y	N	N
40	Saravanan M	38	M	N	Anterior	Y	N	N	Y	N	60	LeftMain	N	N	N	Y	N
41	Baskar	32	M	Y	Anterior	N	N	N	N	N	50	Normal vessel	N	N	N	N	N
42	Muniappan	40	M	Y	inferior	N	N	Y	Y	N	50	Normal vessel	N	N	N	N	N
43	Ellappan	32	M	Y	inferior	Y	N	N	N	N	50	Two vessel	Proximal	N	N	N	N
44	Selvadurai	35	M	N	Nonstemi	Y	N	Y	N	N	55	Normal vessel	N	N	N	N	N
45	Jeyakumar	37	M	Y	Anterior	N	Y	N	N	N	50	Two vessel	Mid	Y	N	N	N
46	Munusamy	40	M	Y	inferior	N	Y	Y	N	N	60	Two vessel	Distal	N	Y	N	N
47	Murugesan	34	M	Y	Anterior	N	N	N	N	N	47	Normal vessel	N	N	N	N	N
48	Thooyamani	39	F	N	inferior	N	Y	Y	Y	N	60	Normal vessel	N	N	N	N	N
49	Rajendran	34	M	Y	inferior	N	Y	N	N	N	45	Normal vessel	N	N	N	N	N
50	Srinivasn	37	M	Y	Anterior	N	N	N	Y	N	48	Two vessel	Mid	N	Y	N	N
51	Murugesan	40	M	Y	inferior	N	Y	N	N	N	48	One vessel	N	N	Y	N	N
52	Ganesan	38	M	Y	inferior	N	Y	Y	N	N	50	Two vessel	N	N	Y	Y	Y
53	Kasi	35	M	Y	Anterior	N	N	N	N	N	48	One vessel	Proximal	N	N	N	N
54	Karuppasamy	32	M	Y	Anterior	N	Y	N	N	N	50	Normal vessel	N	N	N	N	N
55	Pragalatha	32	M	Y	Anterior	N	N	N	N	N	52	Normal vessel	N	N	N	N	N
56	Suresh	35	M	Y	Anterior	N	N	N	N	N	52	Normal vessel	N	N	N	N	N
57	Prameela	35	F	N	inferior	Y	N	N	N	N	60	Normal vessel	N	N	N	N	N
58	Suresh	36	M	Y	Anterior	N	N	N	N	N	44	One vessel	Mid	N	N	N	N
59	Sarangapani	30	M	Y	Anterior	N	Y	Y	Y	N	50	One vessel	Proximal	N	N	N	N
60	Subramani	30	M	Y	Anterior	N	N	N	N	N	50	One vessel	Mid	N	N	N	N
61	Ambica	40	F	N	Anterior	N	Y	Y	N	N	50	Two vessel	Mid	Y	N	N	N
62	Kannadasan	35	M	Y	Anterior	Y	N	N	N	N	50	One vessel	Mid	N	N	N	N

MI - myocardial infarction
F/H - Family History
SHT - Systemic hypertension
DM - Diabetes mellitus
LAD - Left Anterior descending artery
LCX - Left circumflex artery

Hyper - hyperlipidemia

EF - Ejection fraction

RCA - Right Coronary artery

LMCA - Left Main coronary artery

als

MASTER CHART 2 - OLDER AGE GROUP(>= 55 YEARS)

S.No	Name	Age	Sex	Smoker	MI	F/H	Hyperl	DM	SHT	Obes	EF %	Disease Group	LAD	LCX	RCA	LMCA
1	Visalakshi	58	F	N	Inferior	N	Y	N	N	N	60	Normal Vessel	N	N	N	N
2	Sundararajan	64	M	N	Inferior	N	Y	Y	Y	Y	60	Two Vessel	Mid	N	Y	N
3	Madhavrao	69	M	N	Inferior	N	Y	Y	N	N	55	Two Vessel	Proximal	Y	N	N
4	Paranjothi	55	F	N	Anterior	N	Y	Y	Y	N	50	One vessel	N	N	Y	N
5	YusufKhan	58	M	N	Inferior	N	N	N	Y	N	50	Two Vessel	Proximal	N	Y	N
6	Ramadoss	68	M	Y	Nonstemi	N	N	N	Y	N	68	Three vessel	Mid	Y	Y	N
7	Sundramoorthi	67	M	Y	Nonstemi	N	Y	Y	Y	N	47	Three vessel	Proximal	Y	Y	N
8	Ramasamy	70	M	N	Inferior	N	Y	N	Y	N	57	Two Vessel	Mid	N	Y	N
9	Mohamed saleem	56	M	Y	Anterior	N	Y	Y	N	N	50	One vessel	Proximal	N	N	N
10	Sesuraj	58	M	Y	Inferior	Y	N	N	N	N	40	Three vessel	Mid	Y	Y	N
11	Anjali	55	F	N	Inferior	N	N	N	Y	N	55	Normal Vessel	N	N	N	N
12	Abdul gaffoor	56	M	Y	Inferior	N	Y	N	Y	N	60	One vessel	N	N	Y	N
13	Karpagavalli	58	F	N	Inferior	N	Y	Y	N	N	60	Two Vessel	Mid	N	Y	N
14	Arumugam	77	M	N	Anterior	N	Y	Y	N	N	60	Three vessel	Mid	Y	Y	N
15	SivaShunmugham	65	M	Y	Inferior	N	Y	N	Y	N	60	Three vessel	Mid	Y	N	Y
16	KadarBasha	55	M	N	Anterior	Y	Y	N	N	N	55	Normal Vessel	N	N	N	N
17	Dhanush	60	M	Y	Inferior	N	Y	Y	Y	N	56	Two Vessel	Mid	Y	N	Y
18	Thanikachalam	62	M	N	Nonstemi	N	N	N	Y	N	56	Normal Vessel	N	N	N	N
19	Chellappan	55	M	N	Anterior	N	Y	N	N	N	60	Normal Vessel	N	N	N	N
20	Arokiasamy	56	M	Y	Inferior	N	Y	N	N	N	56	One vessel	N	Y	N	N
21	Illias	57	M	Y	Inferior	Y	Y	Y	N	N	45	Three vessel	Distal	Y	Y	N
22	Sivanjothi	62	M	N	Inferior	N	N	N	Y	N	53	One vessel	N	N	Y	N
23	Gunasekaran	55	M	N	Anterior	N	Y	N	Y	N	60	Three vessel	Mid	Y	Y	N
24	Shunmugasundaram	57	M	N	Anterior	Y	Y	N	N	N	60	Two Vessel	Proximal	Y	N	Y
25	Raffiq	57	M	Y	Inferior	N	Y	Y	Y	N	59	Three vessel	Proximal	Y	Y	N
26	Tamim Ansari	58	M	Y	Anterior	N	Y	Y	Y	N	48	Two Vessel	Proximal	Y	N	Y
27	Chinnathambi	57	M	Y	Anterior	N	N	N	N	N	62	Normal Vessel	N	N	N	N
28	Shankar	55	M	Y	Inferior	Y	Y	N	N	N	60	Two Vessel	N	Y	Y	N
29	Ganesan	59	M	N	Anterior	N	Y	Y	Y	N	61	Two Vessel	Proximal	Y	N	N
30	Mari	56	M	Y	Anterior	N	Y	N	N	N	48	One vessel	Proximal	N	N	N
31	Arumugam	58	M	Y	Anterior	Y	Y	N	N	N	53	One vessel	Proximal	N	N	N
32	Ramu	57	M	N	Anterior	N	Y	Y	Y	Y	50	Three vessel	Mid	Y	Y	N
33	Subramanian	58	M	Y	Anterior	N	Y	N	N	N	45	One vessel	Proximal	N	N	N

34	Gururajan	59	M	Y	Inferior	N	Y	N	N	N	49	Two Vessel	Mid	N	Y	N
35	Doss	56	M	Y	Anterior	N	Y	N	N	N	55	One vessel	N	N	Y	N
36	Subramani K	59	M	N	Inferior	N	Y	Y	N	N	45	One vessel	N	N	Y	N
37	Kannan	57	M	Y	Anterior	N	Y	N	N	N	44	Three vessel	Mid	Y	Y	Y
38	Ramamoorthy	60	M	Y	Inferior	N	Y	Y	N	N	42	Three vessel	Proximal	Y	Y	Y
39	Munusamy	65	M	Y	Inferior	N	N	N	N	N	48	Two Vessel	N	Y	Y	N
40	Elumalai	63	M	Y	Inferior	N	Y	N	N	N	55	One vessel	N	N	Y	N
41	Rajeswari	65	F	N	Inferior	N	Y	N	Y	N	50	Two Vessel	MId	N	Y	N
42	Francis	56	M	Y	Anterior	N	Y	N	N	N	55	Three vessel	Proximal	Y	Y	N
43	Antony	73	M	Y	Inferior	N	Y	N	Y	N	44	Three vessel	Mid	Y	Y	N
44	Karpagavalli	58	F	N	Inferior	N	Y	N	Y	N	50	Two Vessel	N	N	Y	Y
45	Natesan	73	M	N	Anterior	N	N	N	N	N	50	One vessel	Mid	N	N	N
46	Rajendaran	59	M	Y	Anterior	Y	Y	N	N	N	48	Three vessel	Proximal	Y	Y	Y
47	Shunmugam	56	M	N	Inferior	N	Y	Y	Y	N	45	Three vessel	Proximal	Y	Y	Y
48	Esakkimuthu	57	M	Y	Nonstemi	N	Y	Y	N	N	60	Two Vessel	Mid	Y	N	N
49	Sheik davood	56	M	Y	Inferior	Y	N	N	N	N	58	One vessel	N	N	Y	N
50	Srinivasan	55	M	Y	Anterior	N	Y	Y	N	N	50	Three vessel	Proximal	Y	Y	Y
51	Krishnan	56	M	N	Inferior	N	Y	Y	N	N	60	Three vessel	Proximal	Y	Y	N
52	Khader	65	M	Y	Inferior	Y	Y	N	Y	N	60	Two Vessel	Proximal	Y	N	N
53	Raja Chidambaram	71	M	Y	Inferior	N	Y	Y	Y	N	55	Three vessel	Proximal	Y	Y	N
54	Balasubramanian	56	M	Y	Inferior	Y	N	N	Y	Y	55	One vessel	N	N	Y	N
55	Kuppusamy	63	M	Y	Inferior	N	Y	N	Y	Y	50	Two Vessel	N	Y	Y	N
56	Srinivasan A	59	M	Y	Inferior	N	Y	N	Y	N	55	Three vessel	Proximal	Y	Y	N
57	Ramasamy S	60	M	N	Anterior	N	Y	Y	Y	Y	50	Three vessel	Proximal	Y	Y	Y
58	Antony Raj	58	M	Y	Inferior	N	Y	Y	N	N	55	Two Vessel	N	Y	Y	N
59	Nazer Mohideen	57	M	Y	Anterior	N	Y	Y	N	N	48	One vessel	Proximal	N	N	N
60	Deenadayalan	56	M	N	Anterior	N	Y	Y	Y	Y	40	Three vessel	Proximal	Y	Y	Y
61	Mani	55	M	Y	Inferior	Y	Y	N	N	N	55	Two Vessel	Proximal	Y	N	N
62	Srishanth	66	M	Y	Anterior	N	Y	Y	N	N	50	Two Vessel	Proximal	N	Y	N
63	Taj	57	M	N	Anterior	N	Y	Y	Y	N	48	Three vessel	Mid	Y	Y	N
64	Arputhasamy	70	M	Y	Inferior	N	Y	N	Y	N	50	Three vessel	Proximal	Y	Y	Y
65	Jaffer sadiq	62	M	Y	Anterior	N	Y	N	Y	N	48	Three vessel	Mid	Y	Y	N
66	Chandran	63	M	Y	Anterior	N	Y	N	Y	N	42	Three vessel	Mid	Y	Y	N
67	Karunakaran	56	M	Y	Anterior	N	Y	N	Y	N	48	One vessel	Proximal	N	N	N
68	Mohana Sundaram	58	M	N	Inferior	N	N	N	Y	N	60	Normal Vessel	N	N	N	N
69	Duraipandi	56	M	Y	Anterior	N	Y	Y	N	N	48	One vessel	Proximal	N	N	N
70	Elangovan	56	M	Y	Anterior	N	N	N	Y	N	48	Three vessel	Proximal	Y	Y	N

71	Pacilaeema	58	F	N	Inferior	N	Y	Y	N	N	55	Three vessel	Proximal	Y	Y	Y
72	Subbammal	65	F	N	Anterior	N	Y	Y	Y	N	48	Three vessel	Mid	Y	Y	N
73	Gobinath	62	M	Y	Anterior	N	N	N	Y	N	46	Three vessel	Mid	Y	Y	N
74	Parthiban	56	M	Y	Anterior	N	Y	Y	N	N	48	Two Vessel	Proximal	N	Y	N
75	Govindarajan	55	M	Y	Inferior	N	N	N	N	N	55	Two Vessel	N	Y	Y	N
76	Thilagaraj	56	M	Y	Anterior	N	Y	N	Y	N	50	Normal Vessel	N	N	N	N
77	Vairakannu	60	M	Y	Anterior	N	Y	Y	N	N	48	One vessel	Proximal	N	N	N
78	Vadivelu	57	M	Y	Inferior	N	N	N	N	N	60	One vessel	N	Y	N	N
79	Balwanthkaur	60	F	N	Anterior	N	Y	N	Y	N	50	Three vessel	Mid	Y	Y	N
80	Kanikaraj	56	M	Y	Inferior	N	Y	Y	N	N	55	Two Vessel	Proximal	N	Y	N

MI - myocardial infarction
F/H - Family History
Hyper - hyperlipidemia
SHT - Systemic hypertension
DM - Diabetes mellitus
EF - Ejection fraction
LAD - Left Anterior descending artery
LCX - Left circumflex artery
RCA - Right Coronary artery
LMCA - Left Main coronary artery

Collaterals
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PROFORMA

RISK FACTOR ANALYSIS AND ANGIOGRAPHIC PROFILE IN YOUNG MYOCARDIAL INFARCTION

Name **Age** **Sex**

History of Chest pain / Dyspnea / Palpitation / Syncope

DM SHT F/H of premature CAD

Smoking BMI Waist Hip Ratio

Thrombolytic therapy: Yes / No

ECG CKMB Lipid profile

Echo

Coronary Angiogram : Percentage of stenosis

Lesion site : LMCA
LAD
LCX
RCA

Collaterals : Present / Absent